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Case Report

A case of dual atrioventricular nodal nonreentrant tachycardia: An unusual cause of tachycardia-induced cardiomyopathy

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ABSTRACT

We report on a 45-year-old female who developed cardiomyopathy due to incessant dual atrioventricular nodal nonreentrant tachycardia. Her condition was completely resolved by performing radiofrequency ablation of the slow pathway.

1. Introduction

A double ventricular response to a single atrial depolarization is described as a dual atrioventricular (AV) nodal nonreentrant tachycardia (DAVNNT), which is a rare form of arrhythmia that is being reported at an increasing frequency. To our knowledge, few cases of this arrhythmia with tachycardia-mediated cardiomyopathy have been reported [1,2]. We describe a 45-year-old female who presented with congestive heart failure and incessant DAVNNT that was completely resolved by performing radiofrequency (RF) ablation of the slow pathway.

2. Case report

A 45-year-old female was referred to our hospital from another center because of symptomatic congestive heart failure and drug-resistant incessant nonsustained supraventricular tachycardia. In the last 6 months, she had aggravated dyspnea on exertion. On admission, she had dyspnea at rest, distended jugular veins, and pulmonary rales. She denied sudden onset palpitation. A dilated left ventricle with an ejection fraction of approximately 30% was detected on echocardiography. Electrocardiogram (ECG) showed frequent supraventricular premature beats or junctional ectopic beats with an average heart rate of 130 beats/min. In the last 3 months, several drugs such as a beta blocker (i.e., sotalol) and digoxin failed to control her arrhythmia and symptoms.

After discontinuing the antiarrhythmic drugs (i.e., the beta blocker and digoxin), the patient was sent to the catheterization room to undergo an electrophysiological study during the arrhythmia. Four catheter electrodes were inserted through the right and left femoral veins, and they were positioned in the high right atrial, right ventricular apex, His bundle region, and coronary sinus.

During sinus rhythm, the atrio-His (AH) and His-ventricle (HV) intervals measured 62 ms and 40 ms, respectively. During incremental atrial pacing, a 2:1 atrioventricular block developed at a pacing cycle length of 220 ms. However, because of frequent double responses, standard evaluation of the AV node with premature atrial extrastimuli could not be performed. During ventricular pacing, retrograde VA conduction was absent.

Three surface ECG leads and intracardiac recordings of spontaneous tachycardia are shown in Fig. 1. A 1:2 AV relationship was noted. A His deflection was visible before every ventricular activity with a constant and normal HV interval. Interestingly, a pattern of group beating was obvious with progressive prolongation of AH1 (fast pathway conduction) and AH2 (slow pathway conduction) that ended with a complete block of the slow pathway conduction, which was compatible with decremental properties of the AV node. The postulated mechanism is suggested in the ladder diagram of Fig. 1.

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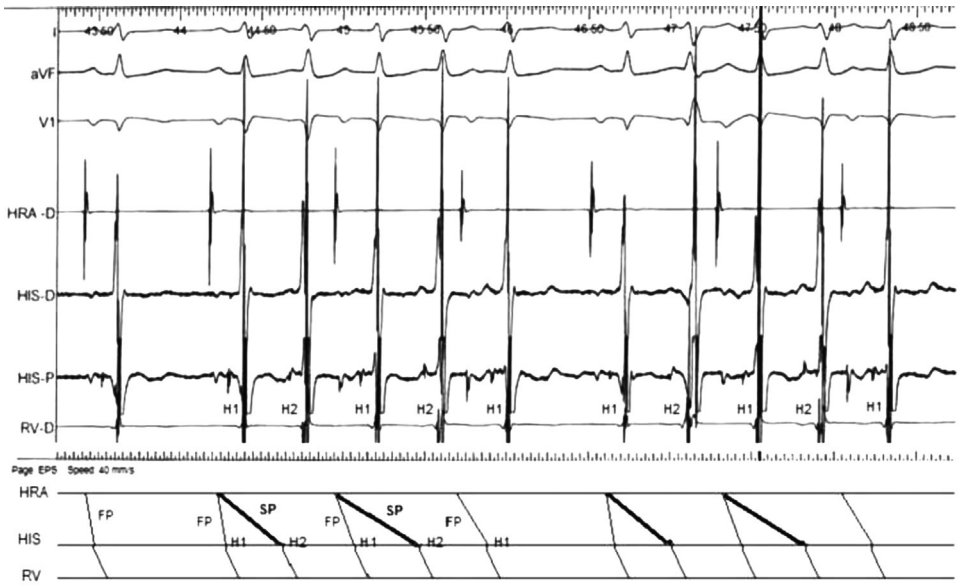

Fig. 1. Simultaneous recording of three surface electrocardiogram leads and intracardiac electrograms from the distal high right atrium (HRA-D), His bundle proximal (HIS-P), His bundle distal (HIS-D), and right ventricular apex (RV-D). In the ladder diagram, the conduction of every atrial signal though the fast and slow pathways, progressive prolongation of the conduction of these pathways, and the Wenckebach-like phenomenon are shown. H1, His bundle activation through the fast pathway; H2, His bundle activation through the slow pathway; FP, fast pathway; SP, slow pathway.

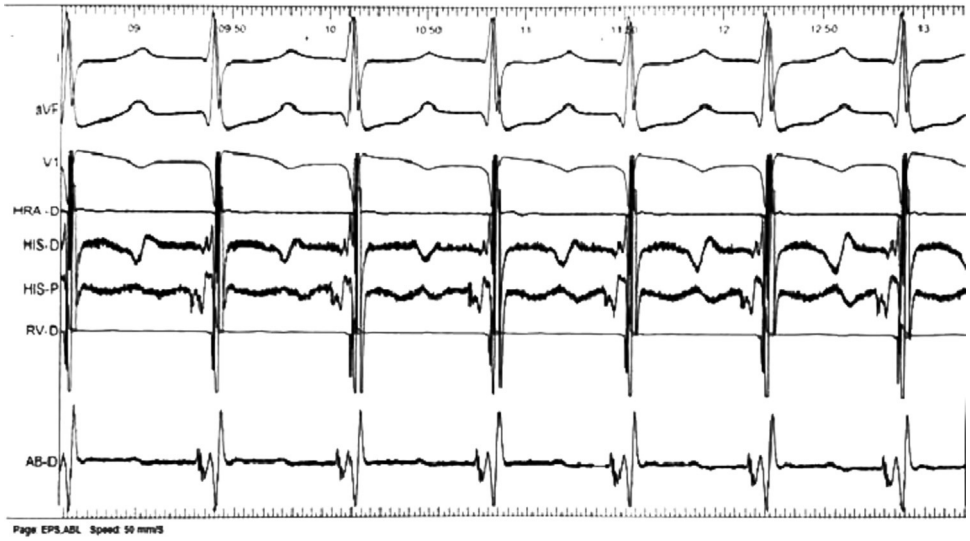

Fig. 2. Induction of the junctional rhythm during the application of radiofrequency to the right atrial posteroseptal at the slow pathway region. HRA-D, distal high right atrium; HIS-P, proximal His bundle; HIS-D, distal His bundle; RV-D, right ventricular apex; AB-D, distal ablation.

A good slow pathway potential and appropriate A/V ratio was obtained via an anatomical approach in the right atrial posteroseptal region. Subsequently, RF application at this site (55 °C, 60 s) resulted in a junctional rhythm (Fig. 2) and the successful elimination of tachycardia and non-inducibility of arrhythmia during the electrophysiology study. During the post-ablation study, no sign of dual conduction was detected. AV conduction was occurring only along the fast pathway, and the effective refractory period of AVN was reached at 330 ms without an AH jump (Fig. 3). The rhythm was sinus with stable PR intervals.

After ablation, the patient was well without symptoms. Her left ventricle ejection fraction improved to 55% within 1 month of follow-up.

3. Discussion

We present a rare case of supraventricular tachycardia based on a dual AV nodal physiology. Although typical and atypical AV nodal reentrant tachycardias are common arrhythmias due to this physiology, DAVNNT is also reported with a nonreentrant pattern based on the existence of a dual AV nodal pathway. A dual ventricular response to a single atrial impulse due to simultaneous fast and slow pathway conduction can be observed during an electrophysiology study (i.e., each ventricular activation is preceded by a His bundle deflection) [1,2]. This arrhythmia is sometimes misinterpreted as atrial fibrillation, and some of these cases were detected in the electrophysiology laboratories when they were referred for pulmonary vein isolation [2].

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Fig. 3. The pacing stimuli are delivered from the distal high right atrium (HRA-D) catheter at a 500-ms cycle length (S1 interval) with a 330-ms S1–S2 interval after slow pathway ablation. No antegrade conduction after premature atrial pacing is observed. HIS-P, proximal His bundle; HIS-D, distal His bundle; RV-D, right ventricular apex; AB-D, distal ablation.

The requirements for establishing this arrhythmia are as follows: (1) a slow pathway (or maybe a fast pathway) should not have retrograde conduction; and (2) the refractory period of the distal common pathway must be shorter than the difference in the conduction times over the fast and slow pathways so that the impulses of these pathways reach the His bundle and ventricles after refractoriness of the previous impulse [2].

The most important differential diagnosis for DAVNNT is Hisian bigeminal extrasystoles. Differentiating between these two phenomena is difficult. In our patient, there were some clues that ruled out Hisian extrasystoles. Retrograde His activation (i.e., activation of the proximal His after the distal His) and shortening of the HV interval are indicative of parahisian extrasystole [3]. However, in our case, His was activated antegradely with an equal HV interval compared to the sinus rhythm (Fig. 1). Prolongation of the AH1 and AH2 interval with a Wenckebach pattern, as shown in our case, is much more compatible with decremental conduction from the fast and slow pathway [4]. These criteria increase the likelihood of dual AV node physiology, but complete disappearance of the arrhythmia after performing slow pathway ablation is the strongest reason for making this diagnosis.

During the basic study, we did not observe any retrograde conduction from the AV node with ventricular pacing. However, during ablation of the slow pathway, the junctional rhythm showed antegrade and retrograde conduction. This means that the fast pathway had antegrade and retrograde conduction in our case. Yet, the pathway under the slow pathway only had unidirectional conduction.

After slow pathway ablation, AV block no longer occurred. This can be explained by two theories. In the case of a lower common pathway, two times conduction of each P wave into the lower common pathway resulted in decremental conduction through this pathway before ablation of the slow pathway. However, post-ablation, as each P wave conduction in this pathway became 1:1, the PR stabilized. The other theory is that with ablation of the slow pathway, the fast pathway conduction was enhanced because retrograde concealed conduction of the fast pathway was eliminated.

Until recently, very rare cases of tachycardia-induced cardiomyopathy due to this arrhythmia have resolved by slow pathway ablation similar to our case [1]. Thus, it seems that the early detection of this arrhythmia and definitive treatment, especially in cases of tachycardiomyopathy, is important to prevent or minimize the effects of persistent tachycardia on cardiac function.

4. Conclusions

Dual atrioventricular nodal nonreentrant tachycardia is a rare arrhythmia that can cause tachycardia-induced cardiomyopathy. Ablation of the slow pathway should be considered as a curative treatment.

Conflict of interest

The authors declare no conflicts of interest.

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References

[1] Wang NC. Dual atrioventricular nodal nonreentrant tachycardia: a systematic review. Pacing Clin Electrophysiol 2011;34:1671–81.
[2] Francis J, Krishnan M. Dual ventricular response or 1:2 atrioventricular conduction in dual atrioventricular nodal physiology. Indian Pacing Electrophysiol J 2008;8:77.
[3] Hadid C, Di Toro D, Gallino S, et al. Differential diagnosis between dual ventricular response and bigeminy arising from the bundle of His. Rev Esp Cardiol (Engl Ed) 2013;66:515.
[4] Maury P, Hebrard A, Duparc A, et al. Incessant non-reentrant supraventricular tachycardia interrupted by apparent simultaneous wenckebach sequences along both nodal pathways. Pacing Clin Electrophysiol 2008;31:757–60.

Please cite this article as: Akbarzadeh MA, et al. A case of dual atrioventricular nodal nonreentrant tachycardia: An unusual cause of tachycardia-induced cardiomyopathy. J Arrhythmia (2015), http://dx.doi.org/10.1016/j.joa.2015.04.008